the triple positivity was significantly associated with male gender (OR=3.5; p=0.02), the presence of rheumatoid nodules (OR=5,3; p=0.015) and pulmonary involvement (OR=2.6; p=0.007). Anti-IFI16 auto-Abs were associated to male gender independently of the presence of the other two auto-Abs.

Conclusions: Our study demonstrated that anti-CEP-1 auto-Abs may participate to the development of RA-associated pulmonary manifestation together with anti-CCP and that the assessment of multiple auto-Abs in daily practice may help clinician to stratify RA patients at identify those at higher risk to develop extra-articular manifestations.

References:

[1] Alunno A et al. Arthritis Care Res (Hoboken) 2016;68(4):440–5.
[2] Montes A et al. Arthritis Rheum 2012;64(10):3102–3110.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.3564

AB0192 SERUM MEASURES OF TYPE I COLLAGEN DEGRADATION ARE SURROGATE MARKERS OF JOINT DESTRUCTION AND PROGRESSION; FIRST STEPS TOWARDS A PROGNOSTIC SCORE

A.C. Bay-Jensen[1], C. Bager[2], A.S. Siebuhr[1], H. Bay Nielsen[2], M.A. Karsdal[1]. *[1]Rheumatology, Nordic Bioscience Biomarkers and Research; [2]Proscion, Herlev, Denmark*

Background: Monitoring of patients with rheumatoid arthritis (RA) requires assessment of biomarkers reflecting disease activity and its progression. There is a need for non-invasive markers for frequent monitoring of disease severity and progression as well as response to therapy.

Objectives: Serological markers together with clinical parameters was tested in a multi-marker model to assess its ability to objectively predict progression of RA.

Methods: Current post-hoc analysis included RA patients from the biomarker substudy of the phase III clinical study LITHE investigating the safety and efficacy of tocilizumab[1–4]. Patients had moderate/severe, active RA. In addition, only patients of the placebo arm and with total sharp score (SHP) recorded at baseline (BL), week 24 (W24) and W52 were included. Progressors were defined as the delta from BL to W24 and W24 to W52. Biochemical markers reflecting tissue turnover (table) were assessed at BL and W16. Associations with structural progression (deltaSHP) were investigated by spearman's r, least squared multivariate and logistic regression. Covariates were CRP, sex, BMI, age, disease duration, DAS-ESR, no. prior DMARDs/aTNF use and SHP/BL. The data were divided into a training and confirmation set; 1) association between markers/W16 and deltaSHP/W52 (n=31 prog./42 non-prog.), 2) association between markers/BL and deltaSHP/W24 (n=33/48).

Results: *The training set.* Eight markers were correlated (R>0.2) with deltaSHP/W52. Of these C1M, PINP, ICTP and MMP3 were predictive for of progression (deltaSHP/W52>0) with ORs of 3.2 [1.3–8.0], 4.0 [1.4–12], 8.5 [2.4–31], and 2.5 [1.3–5.1]; all p<0.01, respectively. A logistic model for prediction of disease progression incorporating C1M, ICTP, disease duration and BMI demonstrated an AUC of 0.77 [0.66–0.86], p<0.01. The model correctly identified 72% of the progressors. textitThe confirmation set: The results were confirmed in the second dataset with an AUC of 0.75 [0.64–0.81], p<0.01. The model correctly identified 65% of the progressors.

Biochemical marker	Description	Biomarker of	Spearman correlation between DeltaSHP/W52 and baseline biochemical marker R>0.2
C1M	MMP-mediated type I collagen degradation	Connective tissue destruction	**0.377**
C2M	MMP-mediated type II collagen degradation	Cartilage degradation	0.088
C3M	MMP-mediated type III collagen degradation	Connective tissue destruction	**0.277**
C4M	MMP-mediated type IV collagen degradation	Basement membrane destruction	0.157
C6M	MMP-mediated type VI collagen degradation	Connective tissue destruction	0.175
CRP	C-reactive protein	Acute reactant	**0.354**
CRPM	C-reactive degradation	Tissue inflammation	**0.290**
CTX-I/OC	Ratio between cathepsin K-mediated type I collagen degradation and osteocalcin	Bone turnover balance (Bone resporption/formation)	0.063
Gender	-	-	0.090
HAQ	Health assessment questionnaire		-0.032
ICTP	MMP-mediated type I collagen degradation	Connective tissue destruction /bone degradation	**0.334**
MMP3	Matrix metalloproteinase 3	Joint inflammation	**0.226**
Pain (VAS)	-	-	0.073
Patient global score (VAS)	-	-	0.110
Physician global score (VAS)	-	-	0.051
PIIANP	Propeptide of type II collagen	Cartilage formation	-0.077
PINP	Propeptide of type I collagen	Connective tissue and bone formation	**0.217**
VCAM	MMP-mediated Versican degradation	Epithelial turnover	-0.054
VICM	MMP-mediated degradation of citrullinated vimentin	Macrophage activity	**0.232**

Conclusions: We demonstrated that a multi-marker model was able to pint-point, which patients were more likely to be structural progressors. Such first steps to build a progression model, rather than a score reflecting disease activity only, may enrich clinical studies with structurally active diseaese. Importantly, the markers with strongest influence were those associated with MMP-driven bone (ICTP) and connective tissue (C1M) remodelling.

References:

[1] Smolen JS.Tocilizumab inhibits progression of joint damage in rheumatoid arthritis irrespective of its anti-inflammatory effects: disassociation of the link between inflammation and destruction. Ann Rheum Dis 2012.
[2] Kremer JM.Tocilizumab inhibits structural joint damage in rheumatoid arthritis patients with inadequate responses to methotrexate: results from the double-blind treatment phase of a randomized placebo-controlled trial of tocilizumab safety and prevention of structu. Arthritis Rheum 2011.
[3] Bay-Jensen AC. Effect of tocilizumab combined with methotrexate on circulating biomarkers of synovium, cartilage, and bone in the LITHE study. Semin Arthritis Rheum 2014.
[4] Bay-Jensen AC. Early changes in blood-based joint tissue destruction biomarkers are predictive of response to tocilizumab in the LITHE study.Arthritis Res Ther 2016.

Disclosure of Interest: A. Bay-Jensen Shareholder of: Nordic Bioscience, Employee of: Nordic Bioscience, C. Bager Employee of: Proscion, A. S. Siebuhr Employee of: Nordic Bioscience, H. Bay Nielsen Employee of: Proscion, M. Karsdal Shareholder of: Nordic Bioscience, Employee of: Nordic Bioscience

DOI: 10.1136/annrheumdis-2017-eular.6676

AB0193 MEDICAL ADHERENCE IN PATIENTS WITH TIGHTLY CONTROLLED RHEUMATOID ARTHRITIS

A. Caliskan Uckun, F.G. Yurdakul, H. Bodur. *Physical Medicine and Rehabilitation, Ankara Numune Research and Education Hospital, Ankara, Turkey*

Background: Medication adherence is very important in the treatment of rheumatoid arthritis (RA). However, medication adherence of the patients with RA was not optimal in many of the studies (1–2).

Objectives: The purpose of this study was to investigate the medication adherence in tightly controlled RA patients and reasons of non-adherence.

Methods: A total of 82 RA patients (65 women and 17 men) who followed regularly in our outpatient clinic were included. Socio-demographic features and medical history were collected. The eight-item Morisky scale (MMAS-8) was used to evaluate adherence to medication. Disease activity score (DAS28), health assessment questionnaire (HAQ), mini mental state examination (MMSE) test and Beck depression inventory (BDI) were evaluated.

Results: According to Morisky scale, 34.1%, 15.9% and 50% of our patients were categorized as low, moderate and high adherence, respectively. The most prevalent noticed barriers for adherence were forgetting medication, inadequate information about using instructions, side effects of medications (Table 1). Socio demographic features, duration of disease, type and number of drugs used per day, the route of drug administration, co-morbid diseases, body mass index, smoking and alcohol consumption were not found to be associated with medication adherence, whereas low MMSE and high BDI score were associated with low medication adherence (p=0.009 and p=0.011, respectively). We found that the disease activity was significantly higher in non-adherent cases (p=0.00) (Table 2).

Table 1. Barriers to medication adherence

Barriers	%
Forgetfulness	41.4%
Inadequate information	22%
Side effects of medications	17%
Fears about drug benefit	12.2%
Anxiety about side effects	4.8%
Cost of medications	2.6%

Table 2. Medication adherence and disease activity

DAS 28	Low adherence (Morisky <6)	Moderate adherence (Morisky 6–7)	High adherence (Morisky = 8)	Total
Remission (<2.6)	2 (5.6%)	5 (13.9%)	29 (80.6%)	36
Low disease activity (2.6–<3.2)	7 (36.8%)	3 (15.8%)	9 (47.4%)	19
Moderate disease activity (3.2–<5.1)	17 (68.0%)	5 (20%)	3 (12%)	25
High disease activity (≥5.1)	2 (100%)	0 (0%)	0 (0%)	2
Total	28	13	41	82

DAS: Disease activity score.

Conclusions: Our RA patients who were closely followed had 50% high medication adherence. This rate is quite high compared to other studies using MMAS-8. It should be kept in mind that tight control and adequate communication increase medication adherence but different parameters may also be effective. Assessing cognitive disorders and emotional problems of the patient will be beneficial for improving adherence and controlling disease activity.

References:

[1] Gadallah MA, Boulos DN, Gebrel A, Dewedar S, Morisky DE. Assessment of rheumatoid arthritis patients' adherence to treatment. Am J Med Sci. 2015;349:151–6.
[2] Ferguson A, Ibrahim FA, Thomas V, Weinman J, Simpson C, Cope AP, Scott